

Shingles

A plain-English guide for you and your family

WHAT IS HAPPENING

After you had chickenpox as a kid, the virus never left—it fell asleep inside nerve bundles next to your spine. Your immune system kept it locked down for years. But when your defenses drop (from getting older, stress, cancer treatment, or other illness), the virus wakes up. It crawls down one nerve like a train on a track, burning the nerve and the strip of skin that nerve controls. You feel burning or tingling first, then a painful rash with fluid-filled blisters erupts in a band on one side of your body—often the chest, belly, or face. The rash crusts over in about a week. The danger is lasting nerve pain (postherpetic neuralgia) or blindness if the eye nerve is involved. Antivirals like valacyclovir must start within 72 hours to stop the virus from scarring the nerve. The Shingrix vaccine (two shots) cuts your risk by over 90% and is recommended for everyone 50 and older, even if you've had shingles before.

What you might feel

- One-sided band or stripe of painful red bumps that turn into blisters
- Sharp, burning, or stabbing pain in the rash area, often starting before the rash appears
- Tingling, itching, or numbness in the rash area
- Fever, headache, and feeling wiped out
- Blisters that crust over after about a week
- Rash on the face or near the eye (ophthalmic zoster)
- Weakness or drooping on one side of the face plus ear blisters and severe ear pain (Ramsay Hunt syndrome)
- Rash spreading to more than one body area or all over the body

Call the doctor right away if...

- Rash or pain near the eye or on the tip of the nose (risks vision loss from eye infection)
- Facial weakness, drooping eyelid, or ear blisters (Ramsay Hunt syndrome can cause permanent nerve damage if not treated fast)

- Rash spreads to many body parts or looks like chickenpox all over (sign of disseminated zoster, meaning the virus is in the blood and the immune system is very weak)
- Severe headache, stiff neck, confusion, or trouble staying awake (could mean the virus has reached the brain or spinal fluid, causing meningitis or encephalitis)
- Pain that does not improve after the rash heals or lasts more than 3 months (postherpetic neuralgia, or PHN — nerve damage that causes chronic pain)

What the care team will do

- Start antiviral medicine (acyclovir 800 mg five times daily, valacyclovir 1000 mg three times daily, or famciclovir 500 mg three times daily) within 72 hours of rash onset
- Arrange urgent eye exam (within 24 hours) if rash involves the forehead, upper eyelid, or tip of the nose
- Begin nerve-pain medicine (gabapentin starting at 300 mg three times daily or pregabalin starting at 75 mg twice daily) for burning or electric-shock pain
- Keep the rash clean and dry; apply loose, non-stick dressings; tell the patient not to scratch or pop blisters
- Isolate patient from pregnant women, newborn babies, and people with weak immune systems until all blisters have crusted over
- Apply topical lidocaine 5-percent patches or cream to the rash area up to three times daily for localized pain relief
- Screen for depression and sleep problems; refer for counseling if severe pain is limiting daily activities
- Schedule Shingrix vaccine two to six months after the acute rash heals (if patient is age 50 or older and has not received it)

Medicines you may be given — and why

Acyclovir (Zovirax)

Acyclovir tricks the varicella-zoster virus (the chickenpox virus hiding in your nerves) into using it as a fake building block when the virus tries to copy itself. Once the virus grabs acyclovir, the copying stops—like a broken key jamming a lock—so the virus can't multiply and spread to more skin cells.

Valacyclovir (Valtrex)

Valacyclovir is a prodrug—it turns into acyclovir once it is absorbed in your gut. The advantage is that your body absorbs valacyclovir much better than plain acyclovir, so you can take fewer pills per day and still get high levels of the active drug. Once it becomes acyclovir, it works

exactly the same way: it jams the virus's DNA-copying machine so the varicella-zoster virus cannot multiply.

Famciclovir (Famvir)

Famciclovir is another prodrug that your body converts into penciclovir, which works just like acyclovir: it stops the varicella-zoster virus from copying its DNA. Famciclovir is absorbed well and stays active in infected cells for a long time, so it can be dosed less often. It jams the virus's copying machine so the virus cannot make new particles.

Acetaminophen (Tylenol) or Ibuprofen (Advil, Motrin)

Acetaminophen lowers pain signals in the brain and reduces fever; the exact spot it works is still debated, but it does NOT stop inflammation in the skin. Ibuprofen blocks an enzyme called cyclooxygenase (COX) that makes prostaglandins—chemicals that ramp up pain, heat, and swelling at the rash site. By cutting prostaglandin production, ibuprofen dials down both pain and inflammation.

Gabapentin (Neurontin)

Gabapentin calms overexcited nerves by binding to a calcium channel (a gate on nerve cells that controls how much calcium flows in). When gabapentin plugs this gate, less calcium enters the nerve ending, so the nerve releases fewer excitatory signals (like glutamate). This quiets down the constant pain signals firing from damaged nerves in shingles, especially the burning, shooting, or electric-shock sensations.

Pregabalin (Lyrica)

Pregabalin works almost identically to gabapentin: it binds to the alpha-2-delta subunit of calcium channels on nerve endings, reducing calcium influx and cutting down the release of excitatory neurotransmitters like glutamate. This quiets the damaged nerves that are firing pain signals after shingles. Pregabalin is absorbed more predictably than gabapentin, so the dose can often be increased faster.

Lidocaine 5% patch (Lidoderm) or topical lidocaine gel

Lidocaine blocks sodium channels on nerve endings in the skin. Sodium channels are gates that let sodium ions rush into the nerve, which creates the electrical signal that says 'pain.' When lidocaine plugs these gates, the nerve cannot fire, so pain signals from the rash area never start. The patch delivers lidocaine only to the skin, so very little gets into the bloodstream—making it safe and free of sedation.

Opioid analgesics (e.g. oxycodone, tramadol)

Opioids plug into opioid receptors (mu, delta, kappa) in the spinal cord and brain. These receptors are part of the body's natural pain-control system. When an opioid binds, it tells the

nerve cell to release less substance P (a pain chemical) and to open potassium channels (which calm the nerve down). The result: pain signals are muffled before they reach the brain's pain centers, and the emotional weight of the pain (how much it bothers you) also drops. Opioids are reserved for severe shingles pain that other medicines do not control, and only for short-term use.

Capsaicin 8% patch (Qutenza) or lower-strength cream

Capsaicin is the chemical that makes chili peppers hot. It works by overstimulating pain nerve endings that have a receptor called TRPV1. When you first apply capsaicin, it causes intense burning because it floods these nerves with signals. But after a few hours to days, the nerves run out of substance P (the neurotransmitter that carries pain signals) and become temporarily numb. The high-dose 8% patch is applied once in a clinic (under local anesthetic) and can reduce nerve pain for months. Lower-dose creams (0.025%–0.075%) are applied at home several times a day, and they work more gradually by depleting substance P over weeks.

Where this information comes from

- CDC — Shingles (Herpes Zoster) Clinical Overview
- ACIP (Advisory Committee on Immunization Practices) — Prevention of Herpes Zoster: Recommendations, 2018
- American Academy of Dermatology — Shingles Diagnosis and Treatment Guidelines
- Cochrane — Antiviral Treatment for Preventing Postherpetic Neuralgia

Last reviewed: 2026-07-07